LIMITED AREA OF ANESTHESIA, EPILEPTIFORM ATTACKS OF HEMIALGESIA, AND EARLY MUSCULAR ATROPHY IN A CASE OF BRAIN TUMOR.[1]

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The following case is reported because showing as result of a cerebral lesion: (1) circumscribed limited area of anesthesia; (2) marked muscular atrophy, appearing early; (3) epileptiform attacks of hemialgesia of a peculiar nature; (4) loss of the muscular sense, astereognosis, ataxia, and paresis, increasing to ultimate paralysis.

Mary S., age 19, appeared at the Neurological Department of the Boston City Hospital in November, 1902. She reported that she had been perfectly well until within about a year, when she had had some pain in the right shoulder which had promptly yielded to treatment. Six months later this pain in the shoulder returned, and she had suffered from recurring attacks of it since that time. On questioning her it became apparent that the attacks were of a somewhat peculiar character: beginning in a definite region of the body and well defined, the pain spread to successive segments of one side of the body until the whole right side became involved, much as in epilepsy the spasms may extend to adjoining muscular groups. The attacks, which always affected the same side, lasted about half an hour. I had an opportunity to observe some of these attacks. The pain began in the fingers of the right hand, extended up the arm and around the shoulder to the back of the neck, and then involved the right side of the face, the inside of the mouth, the tongue, and the eye of the same side. Having reached this stage, the pain began at once to extend down the back and the right leg, eventuating in a right hemialgesia. The patient stated that these attacks had lately come on every week or two, and that in some of them she had lost consciousness. There was a history also—confirmed by her physician—of attacks of clonic spasm in the right arm, accompanying the pain.

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[1] Read in abstract at the meeting of the American Neurological Association, June 4 and 5, 1906.

In one or two of the attacks of pain which I had the opportunity of observing, there was evidently some disturbance of consciousness, the patient being unable to answer questions, but consciousness was not absolutely lost. Clearly, the attacks were of an epileptiform character.

During much of the time there had been a good deal of headache and dizziness, especially with the attacks of pain. Nausea also had accompanied the attacks, but had not been present at other times. There had been no vomiting. In May preceding her first appearance at the hospital, that is at the time these attacks became recurrent, her physician called her attention to the fact that there was wasting of the shoulder muscles of the right arm. Since that time the arm has been steadily growing weaker.

Examination. Considerable general weakness in the right upper extremity: she could scarcely hold her arm out straight, but she had good control of all the muscles, including those of the hand. There was marked wasting of all the arm muscles, evident also in the small muscles of the hand: the deltoid and possibly the outer head of the triceps were most prominently affected. The atrophy was out of proportion to the paresis and sufficient to suggest spinal origin, but the electrical reactions of the affected muscles were perfectly normal. Mechanical irritability was increased in the muscles of the right arm, but no fibrillary twitching was observed.

The deep reflexes of the right arm were somewhat exaggerated and greater than those of the left. The knee-jerk was increased on the right but not on the left. There was no ankle clonus and no Babinski. Corresponding to the increased knee-jerk, the patient complained that the right leg felt weaker than the left.

Tactile sensation was slightly diminished over a circumscribed area, which included the right shoulder, the right side of the neck, and the right side of the face. The thermal sense was normal, and the pain sense was not diminished out of proportion to the tactile sense. Thus it will be seen that there was no sensory dissociation, such as is characteristic of syringomyelia.

Examination of the eyes showed the presence of optic neuritis.

A diagnosis was made at this time of cerebral tumor.

Shortly afterward the patient left the hospital, against advice, and was not seen again for three months, when (February 20, 1903) I visited her at her home. Her condition had changed very markedly for the worse. She was absolutely blind, and was unable to walk on account of paralysis of the right leg. The weakness of the right arm was much increased, so that she could not move the fingers at all and could scarcely raise the arm at the shoulder. There was slight flexion and extension at the elbow. The paralysis was of flaccid type. Atrophy of the hand

and arm was more marked and fairly symmetrical. The condition of sensation was very interesting. Loss of tactile sense was slight and about the same in intensity as at the first examination, but it had extended over the whole right arm. She could feel a very slight touch of the examining finger, but not so well as on the left. Pain sense, tested with a pin, was normal, but the muscular sense of the fingers was so much impaired as to be nearly lost; she could not tell with any certainty the position of her fingers, whether flexed or extended. Heat and cold she recognized. In contrast with the slight loss of tactile sense and corresponding with the great loss of the muscular sense, there was complete astereognosis. The patient could not recognize any object put into her hand, even when her fingers were pressed down upon it. (Sensation over the remainder of her body was not tested at this time.) In regard to her previous symptoms, she stated that since the blindness had come on, the headache and epileptiform attacks had ceased; also, that she had had much nausea but little or no vomiting.

On March 6, about a fortnight later, she was again examined and her condition found to have grown progressively worse. The whole right upper extremity was absolutely paralyzed. The lower extremity was paretic, but she could move it about the bed. There was a slight weakness of the right side of the face. There was some contracture of the bicipital group, and the fingers were flexed into the palm by a similar contracture; the fingers and arm could, however, be partially extended. There was also slight resistance to passive motion of the right leg as compared with the left (i. e., slight spasticity). On account of mental dulness it was not possible to determine sensation with accuracy. A sharp pin prick could be distinctly felt over the right arm, leg and face. It was not possible to determine astereognosis at this time. Examination for the Babinski sign showed that it could be brought out on both sides, on the right somewhat more markedly. There was no ankle clonus. The right knee jerk was distinctly increased and greater than the left, which was also slightly plus. The deep reflexes of the right arm were slightly greater than the left, but were not excessive. The whole right arm was smaller than the left. Marked atrophy was present in all the muscles. There was very little difference in the lower extremities. With faradism there was no perceptible difference between the reactions of the small muscles of the right hand and those of the muscles of the left, or between the reaction of the right and left deltoid muscles. The same was true also of the galvanic reactions of the same muscles; no polar or other change of R. D.

The diagnosis was that of cerebral tumor, located primarily in the cortical arm area, probably centralized in the post-central convolution, and from there extending backwards into the parietal region and forward into the anterior central convolution. In the hope of relief, an operation was recommended. She was removed to a private hospital and placed under the care of Dr. John C. Munro and operated upon.

The location of the tumor was placed primarily posterior to the fissure of Rolando, on the basis of Sherrington and Grünbaum's experiments (at the time relatively recent). In deference to Mills's views on astereognosis, it was thought that the growth probably had extended backwards into, or exerted pressure backwards upon the parietal region. The secondary developing paralysis, it was thought, could be accounted for by extension or pressure forward into the precentral gyrus. A diagram which I have here and will exhibit, of this probable location, involving chiefly the postcentral convolution, was made just previous to the operation. On removal of the bone flap, the actual site of the tumor, as far as one could tell, corresponded with this diagrammatic localization. The tumor was removed, but, as there was no autopsy, it is impossible to define the exact limits within which the brain was invaded or destroyed. Previous to the operation, the Rolandic fissure was carefully mapped out by two methods, and the bone flap (see report by Dr. Munro) was so made that this fissure traversed it as a central line. The tumor was found about the middle of the opening, and must have involved both of the central convolutions. It was a soft, friable mass, which was found by the pathologist, Dr. Joseph H. Pratt, to measure about 6 cms. in diameter and from 1 to 2½ cms. in thickness. There was no evidence that it had extended far into the parietal lobe, but, owing to the absence of autopsy, the difficulty in determining the exact size of such a friable tumor, its exact location, or the extent of the brain damage, I do not think the case can be cited as evidence of localization of function; though it is neither better nor worse than many cases that have been offered for that purpose. All that can be said is that the findings, taken in connection with the history of the case, are compatible with the theory that the postcentral convolution is sensory, including both the tactile and muscular senses, and possibly stereognosis.

The interesting points are: The original limitation of the (tactile) anesthesia to the neck and side of the head, conforming to the view that there is a focal localization of sensation in the cortex, as with movements; the slight degree of its intensity; the peculiar attacks of pain, spreading by segments; and the muscular atrophy. The last, in the beginning, was out of proportion to the paralysis.*

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*For a digest of the literature on atrophy in cerebral disease see article by Dr. Theodore H. Weisenburg, "A Clinical Study of Hemiplegia in the Adult;" Journal of the American Medical Association, February 25, 1905.

REPORT OF OPERATION BY DR. J. C. MUNRO

On March 10th an osteoplastic operation was performed. An omega-shaped flap (about 2½ in. by 2 in.) was made over the left motor area with its center over the fissure of Rolando and its upper margin about ½ in. from the sagittal suture.

As the flap was turned down it was noticed that the bony plate was eroded probably by pressure from within. The dura was tense, and at the upper medial portion so thin that it was torn by the jaws of the forceps or else had become eroded.

The condition of the patient by this time was so bad that the operation was stopped and the flap replaced and sutured, allowing for a rubber tissue drain.

The patient rallied fairly well in a day or two, and on March 17 the operation was continued.

The bone flap was turned back, and then the dura, when a translucent, pale yellow gelatinous material bulged forth. With the finger a mass of new growth, gliomatous in appearance could be shelled out from the brain, which apparently was pushed away from the dura by the growth. The latter, in places, was apparently incorporated within the brain itself, and some of the growth at this operation was probably left behind. Sharp hemorrhage from the edge of the dura and pia and less from the brain substance itself was controlled by forceps and gauze pressure. The pulse became very weak and rapid (180) and patient was in bad condition.

Venous injection of salt-solution gave some improvement. Gauze packing was pressed between the brain and bone flap and patient put to bed. Under moderate stimulants, she improved considerably, although the pulse continued at 120 and the temperature about normal.

On March 23rd a large hernia had developed, pushing up the flap like a lid and bulging outwards. There was steady leakage of fluid and pus from two stitch holes of the first operation, and about the edge of the flap.

The flap was turned back again and a lot of soft brain removed nearly to the level of the skull. So far as could be told, all the remaining growth and a good deal of infected and healthy brain tissue was removed. There was good pulsation and no evidence of intracerebral pressure.

There was no shock from this operation, and for a day or two she did fairly well. On the 26th her temperature and pulse started to rise, and she failed rapidly, dying on March 28th.

It is unfortunate that the wound infection at or after the second operation could not have been avoided. The enforced delay of a week on account of the bad general condition, the ingrowth of hair with its accompanying seborrhea bathed in cerebrospinal fluid, together with a hurried cleansing at the second operation, all tended to some infection that in a patient of ordinary physical vigor would not have given trouble.

"Specimen consists of soft, opaque, white tissue forming a mass about 6 cm. in diameter. The overlying pia mater is smooth and normal in appearance aside from injection. The surface is nodular. The thickness of the mass is 1 to 2½ cm. on section no gray matter discernible. It is infiltrated with areas of hemorrhage. When hardened in formalin, the tissue is semiopaque and granular on section.

"Microscop. section: The tumor is composed of closely packed cells with round and vesicular nuclei, the cell outlines are not visible. It is very vascular. The cells have a perivascular arrangement and appear to form the outer portion of the vessel's wall. No neuroglia fibers can be demonstrated. Diagnosis: Endothelioma originating probably from the pia mater."